WHAT IS CLAIMED IS:

1. A method of promoting fatty acid oxidation and weight loss in an individual, comprising the step inhibiting the activity of acetyl-CoA carboxylase 2 in said individual.

2. The method of claim 1, wherein said activity is inhibited by administration of an inhibitor of acetyl-CoA carboxylase 2 (ACC2) to said individual.

3. The method of claim 1, wherein said individual has a pathophysiological condition.

4. The method of claim 3, wherein said pathophysiological condition is selected from the group consisting of obesity and diabetes.

5. A method of decreasing blood sugar in an individual, comprising the step of administering an inhibitor of acetyl-CoA carboxylase 2 (ACC2) to said individual.

6. The method of claim 5, wherein said individual has diabetes.

7. A transgenic mouse, said mouse comprising a mutation in an endogenous ACC2 gene for the acetyl-CoA carboxylase 2 isoform of acetyl-CoA carboxylase, wherein said mutation inactivates said gene and results in the lack of expression of a functional acetyl-CoA carboxylase 2 isoform.

8. The mouse of claim 7 wherein one or more exons of said ACC2 gene has been deleted.

9. The mouse of claim 8, wherein said exons have been replaced with heterologous DNA sequences.

10. The mouse of claim 9, wherein said heterologous DNA sequences comprise an hypoxanthine phosphorylribosyltransferase expression cassette.

11. The mouse of claim 10, wherein an exon encoding a biotin binding motif of ACC2 is replaced with an hypoxanthine phosphorylribosyltransferase expression cassette.

12. The mouse of claim 7, wherein said mouse exhibits a phenotype comprising a metabolic reduction in malonyl-CoA production in skeletal muscle and heart.

13. The mouse of claim 12, further comprising a phenotype of unrestricted fat oxidation and reduced fat accumulation in the liver and fat storage cells.

14. The mouse of claim 13, further comprising a phenotype of consuming more calories than a wild-type mouse, yet accumulating less fat than a wild-type mouse.

15. A method of screening for an inhibitor of acetyl-CoA carboxylase 2 isoform activity comprising the steps of:

administering potential inhibitors to wild-type mice; and,

screening for mice which exhibit the phenotype of the transgenic mouse of claim 14.

16. An acetyl-CoA carboxylase 2 inhibitor identified by the method of claim 15.

17. A pharmaceutical composition comprising the acetyl-CoA carboxylase 2 inhibitor of claim 16 and a pharmaceutically acceptable carrier.

18. A method of obtaining a purified preparation of acetyl-CoA carboxylase 1 protein which is free of acetyl-CoA carboxylase 2 comprising the step of:

purifying said acetyl-CoA carboxylase 1 protein from tissues obtained from the transgenic mouse of claim 7.

19. A method of obtaining murine antibodies against acetyl-CoA carboxylase 2 which are less crossreactive with acetyl-CoA carboxylase 1 and other mouse proteins comprising the step of:

generating said antibodies in the transgenic mouse of claim 7.

20. A cell line derived from the transgenic mouse of claim 7.

21. The cell line of claim 20, wherein said cell line is derived from cells selected from the group consisting of muscle cells, heart cells, adipose cells, and liver cells.

22. A method of screening for agonists and antagonists of ACC2 comprising the steps of:

administering a candidate compound to the cell line of claim 20 and to cell lines derived from wild-type mice; and,

monitoring said cell lines for alterations in cellular activity, wherein a compound that specifically acts on ACC2 will have altering cellular activity in wild-type cells but will have no effect on the cell line of claim 20.

23. The method of claim 22, wherein monitored cellular activities are selected from the group consisting of mRNA expression, protein expression, protein secretion, and lipid metabolism.